molecular layer. 2. The layer of small pyramids. 3. Layer of medium sized pyramids. 4. Stellate layer or layer of Gennari. 5. Layer of small spheroidal cells. 6. Layer of giant pyramids. 7. Layer of polymorphous cells. 8. White substance. These layers are again divided, but the original should be consulted. In brief, the outer three layers are more or less typical. The layer of Gennari presents three structures: large stellate cells, large ascending fibers, which, by their rich ramifications, make up the horizontal striations of this layer, and axonal and collateral ascending fibers from the stratum below.

The layer of polymorphous cells consists of at least four types of cells. JELLIFFE.

PATHOLOGY.

154. ZUR HISTOLOGIE DER POLIOMYELITIS ANTERIOR CHRONICA (Histology of Chronic Anterior Poliomyelitis). Max Bielschowsky (Zeitschrift für klin. Med., Vol. xxxvii, Nos. 1 and 2, p. 1).

The patient described in this paper was seventeen years old. He was said to be as healthy as normal boys until his ninth year. At that age, without known cause, a flaccid atrophic paralysis began, and later involved the entire musculature of the extremities and trunk. The paralysis was first noticed in the lower limbs in the peroneal group. In the later stages of the disease the muscles of the neck were affected and the tongue was atrophied and paretic. The paralysis in the beginning of the disease preceded the atrophy; later this mode of involvement was not so noticeable, but the paralysis was always more pronounced than the atrophy. Contracture and kyphoscoliosis developed in the course of the disease. The diagnosis of chronic poliomyelitis was made. The contractures, which were of an extreme type, were not spastic but paralytic, and developed later than the atrophic paralysis. The tendon reflexes were absent and the paralyzed muscles were flaccid. The motor cells presented marked quantitative and qualitative changes throughout the cord as high as the hypoglossal nucleus, and the anterior horn cells which are nourished exclusively by the central artery, were most diseased, while those near the periphery of the anterior horn, which receive some nourishment from the peripheral arteries, were not quite as much diseased. Cells other than motor in the anterior horns were also affected. The nerve fibers and neuroglia fibers of the anterior horns were diminished in number; the intramedullary portions of the anterior roots were much degenerated; the vessels of the anterior horns were diseased and new vessels were formed; round cell infiltration, recent and old hemorrhages were seen in the anterior horns. The lateral pyramidal tract showed no degeneration except by the Marchi method, and even by this method the degeneration was insignificant.

SPILLER.

155. STUDIES ON GANGLION CELLS. J. Ewing (Archives of Neurology and Psycho-pathology, I, 1898, p. 283).

Ewing here gives, in a monograph of 180 pages with six plates, the most comprehensive contribution to ganglion cell pathology yet published. For wealth of material, character of research, bibliographical completeness, excellence of typography and high class illustrations, this monograph is exemplary.

He first discusses the subject of technic and gives a full bibliographical summary and advises the use of alcohol, 97 per cent.; alcohol and corrosive sublimate; formalin sublimate; Van Gehuchten fluid and formalin, 10 per cent, as of about equal service, especially when small pieces of tissue are used. The author's views with reference to the histology of the cell is in close accord with those of Dogiel and Held, but his interpretations are wider than those of most authors. The physiological conditions and the general variations in structure of the ganglion cells in cases dying of hemorrhage and shock, and cadaveric changes in ganglion cells are fully discussed. With reference to the last the opinion is expressed that there is no evidence to show that cadaveric changes can simulate those produced by disease processes. Cell changes in diseases of the nervous system are then taken up, the observations of recent authors being corroborated and amplified. Ewing does not believe that it has been demonstrated that primary and secondary affections of the neuron in the neuritic process can be clearly differentiated by any method at our command at present. The literature on myelitis, Landry's paralysis, tabes, bulbar palsy, paresis and the insanities and idiocy are reviewed, but no original observations are recorded. Investigation of two cases of carbolic poisoning showed an absence of clearly defined lesions, notwithstanding grave nervous symptoms, and in three cases of eclampsia few changes were noted. Six cases of uremia showed marked alterations in the chromatic structures, irregular in character and distribution. The medullary nuclei, notably the tenth nucleus, were most involved. Three cases of sunstroke and numerous cases of hyperpyrexia in rabbits lead the author to believe that in this condition the alterations are always pathognomic in character. In various forms of meningitis the cell changes seemed most marked in the compressed and anemic areas, and led the author to infer that grave circulatory disturbances produce more cellular modifications than soluble toxic materials. In two cases of typhoid, marked cellular lesions distributed throughout the nervous system were observed, probably influenced largely by the hyperpyrexia. Acute lobar pneumonia may or may not induce cellular lesions; all the cases here recorded, three in number, showed some involvement of the medullary nuclei. In cases of general sepsis from bronchitis, pyemia, empyema, phlegmonous inflammation, and tubercular infection, wide spread in character, chromatic alterations were observed in each case. The severity of the cellular lesions seemed in proportion to the amount of toxemia and the temperature. Tetanus and hydrophobia showed changes similar to those described by other writers. Ewing then discusses the general significance of the Nissl bodies and the bearing of them on nutrition and allied conditions making a complete summary of the recent literature. He also reproduces a number of the experiments of Goldscheider and Flatau on hyperthermia, and his results in the main agree with those of these investigators. The writer's general conclusions of the general characters of the lesions in nerve cells is worthy of quotation *in extenso*, but space compels abstracting. He believes that in the general process of chromatolysis the first change is that of a preliminary swelling of the chromatic body, best shown in the cases of tetanus and hyperpyrexia in the early stages. A uniform diminution then results, to be followed by a uniform subdivision of the chromatic bodies, such changes being observed in the chronic toxemias. Granular subdivision of the chromatic bodies may result as a further stage from sudden acute lesions. The final stage in the process, in which the cell body has lost all of its stainable substance, is the "simple complete chromatolysis." This result seems to follow a variety of causes and no hint as to the origin can be gained from the pathological picture. Minute variations in the chromatolytic process appear in nearly every case and any attempt to exhaust all of the possible peculiarities is held to be futile. "The minute lesions appear to differ in every case and the range of minor peculiarities is practically limitless," are the words of the author. When acute degeneration, in a strict sense, affects the ganglion cell, its changes are evidently manifested by various forms of chromatolysis, but the true degenerative process may not, and as the writer believes, "frequently does not begin until after chromatolysis is complete." JELLIFFE.

156. LA TOXICITÉ DE LA SUEUR CHEZ LES EPILEPTIQUES ET LES MÉLANCOLIQUES (Toxicity of the Sweat of Epileptics and in Melancholia). Dr. Mavrojannis (Revue de Psychiatrie, 2, 1898, p. 199.)

Cabitto's researches on the sweat in epileptics were so startling, not to say apocryphal, that it is not surprising to find that his conclusions have been called in question, and, according to the author's experiments, disproven. In a series of experimental injections in rabbits of post-paroxysmal perspiration the results were almost negative. In a few cases there were muscular reactions, but these could be explained on other grounds. The secretions from cases suffering from melancholia were also nontoxic. [A few more such conscientious experiments would probably modify the Bouchard methods, in which connection the recent work of Putnam-Pfaff is to be consulted as a model of careful research.] JELLIFFE.

157. ANCORA SULLA GLIOSI CEREBRALE NEGLI EPILETTICA (Cerebral Gliosis in Epilepsy). A. Tedeschi (Policlinico, 6, 1899, No. 1).

In a recent case of epilepsy the author describes a marked asymmetry of the two hemispheres. The left side was smaller than the right and showed a peculiar structure. All of the structural elements were much simplified, but there was a marked increase in the neurolgia, making a distinct felt-like tissue with few nerve cells and many fibers. There were no alterations in the vessels nor in the meninges. The nerve cells of the right hemisphere were normal, as were the ganglion cells of the basal ganglia, though the left half had fewer elements. There were fewer Purkinje cells in the left half of the cerebellum. JELLIFFE.

158. OBDUCTIONSBEFUNDE BEIM TOD IM STATUS EPILEPTICUS (Autopsy findings in Status Epilepticus). L. W. Weber (Wien. med. Wochenschrift, 1899, p. 157).

Weber has given one of the best microscopical studies of the nervous system in severe epilepsy that has been contributed of recent years. His results were drawn from a series of autopsies with microscopical examinations carried on for the past four years in the asylum of Uchtspringe. The most constant lesions were found in the blood vessels. These in the brain and cord and medulla were found to be filled with blood and markedly dilated. In places there were blood infractions with hyaline degeneration of the walls of the capillaries. In many cases there were extravasations of red blood cells into the lymph spaces and perigangliar spaces. In many of these extravasated areas the blood cells had undergone retrograde changes, and in some instances destructive lesions of the ganglion cells were observed. The ganglion cells of the spinal cord were often found to be rich in pigment, and the vascular lesions were of the same type. The author holds that these vascular degenerations are secondary rather than primary, and looks for the primary cause in some form of endogenous poisoning. The transitory paralyses and pareses, he thinks, are to be attributed to these minute lesions, but the epileptic attack itself is the result of some form of autointoxication. When the lesions involve the cranial nerves death may result. JELLIFFE.